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INVITED COMMENTARY

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Endoleak, the Achilles heel of endovascular aortic aneurysm repair (EVAR), is correlated with aneurysm sac expansion, the need for conversion, aneurysm rupture, and death. The presence of endoleak is the most common reason for readmission to the hospital after EVAR and increases the secondary procedure rate, cost, and length of stay.

Collateral vessel endoleaks (type II), the most prevalent form of endoleaks, do not behave as a uniform class even though they share a common etiology—back bleeding from an aortic branch. As Silverberg et al and others have demonstrated, some of these leaks will spontaneously thrombose, and some will persist. Some will transmit systemic pressure to the aneurysm sac and to lead to rupture of the aneurysm, whereas in other cases, the aneurysm sac regresses and the patients seem to be protected from aneurysm rupture despite the presence of an endoleak. Unlike type I and type III endoleaks, which mandate repair upon their discovery, there is no clear consensus on how type II endoleaks should best be treated, or even monitored.

Why are type II endoleaks so different and unpredictable in their behavior? Vascular surgeons readily understand that type II endoleaks could not remain patent if only supplied by a single vessel, because end arteries with no outflow rapidly thrombose. This explains why most type II endoleaks that are detected immediately postoperatively have disappeared by the time of the first postoperative computed tomography (CT) scan. For a branch vessel endoleak to remain patent, it must have both inflow and outflow. Duplex Doppler ultrasound criteria, which have been found to be predictive of type II endoleak thrombosis, include a high-resistance type of flow, whereas what is most predictive of continuation of the endoleak is a low-resistance, continuous type flow, indicative of a patent outflow tract. The flow patterns in these endoleaks are variable, changing with blood pressure, position, respiration, and other dynamic factors.

This changing flow pattern presents a great challenge to current imaging modalities and explains the frequent observation of "intermittent appearance" of a type II endoleak. CT angiography (CTA) has been shown repeatedly to lack specificity for determination of leak type and vessel origin of type II leaks compared with arteriography or direct sac injection. Injection of the sac in patients who have been found to have a type II endoleak often reveals multiple pairs of lumbar arteries in communication with each other as well as with other branch vessels, when only a single vessel was suspected by the screening CTA images.

The emerging understanding of these endoleaks is a picture analogous to our view of arteriovenous malformations. They are associated with multiple vessels, which may serve as inflow or outflow, depending on the prevailing physiologic state at the moment. The involved vessels share a nidus of communication that maintains the patency of the type II endoleak.

Treatment of type II endoleaks should focus on disruption of this nidus, resulting in end arteries without the possibility of outflow, leading to thrombosis. For this reason, it is no wonder that type II endoleaks related to patent lumbar arteries developed in all 12 (100%) of the patients Silverberg et al treated by means of preoperative inferior mesenteric artery (IMA) embolization! Their prophylactic strategy addressed individual feeding arteries rather than the root cause of the endoleak, which is the communication between multiple arteries. If a prophylactic approach to type II endoleak is to be developed, the greatest likelihood for success would likely be to focus on obliteration of the sac and the paths between branch vessels.

Silverberg et al have taken a mostly selective approach to intervention for type II endoleaks, with the notable exception of the previously mentioned preoperative IMA embolizations, generally reserving intervention for patients who demonstrate significant sac expansion. They have shown that in the short term (22 months' mean follow-up), this approach has not resulted in any aneurysm ruptures. Because they did not apply any standard protocol to the treatment of type II endoleak, and in the absence of a control group for comparison, it is difficult to determine if the behavior of their endoleak patients is different from that of EVAR patients in general or even from the natural history of untreated aneurysms over a 22-month mean follow-up interval. No clear treatment recommendations emerge.

What is clear is that our understanding of type II endoleak and its treatment is evolving. Patients remain at risk for development of endoleaks at all times after EVAR. This is an absolute mandate for continued, careful surveillance of patients who have undergone EVAR.